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1: Eur J Med Res. 1997 Nov 28;2(11):465-8. Related Articles, Links

Fibroblast growth factor-1 prevents myocardial apoptosis triggered by ischemia reperfusion injury.

Cuevas P, Reimers D, Carceller F, Martinez-Coso V, Redondo-Horcajo M, Saenz de Tejada I, Gimenez-Gallego G.

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BACKGROUND: Apoptosis is a constant feature of reperfusion injury in ischemic cardiac myocytes, leading to late cell death. Since fibroblast growth factors (FGFs) inhibit apoptosis in differentiated cells, we hypothesized that FGF-1 (acidic FGF), in its native form, and a non-mitogenic isoform would attenuate myocardial ischemia-reperfusion- induced apoptosis. METHODS AND RESULTS: The effect of native and non-mitogenic fibroblast growth factor-1 mutein (FGF-1 and m-FGF-1) on apoptosis assessed by terminal deoxynucleotidyl transferase-mediated dUTP nick end-labeling (TUNEL) method was tested in a rat model of 20 min regional myocardial ischemia and 24h reperfusion. Myocardial ischemia followed by reperfusion resulted in a high myocardial apoptosis rate in the area at risk. When given as a systemic bolus inmediately after myocardial ischemia, both FGF-1 and m-FGF-1 significantly reduced apoptosis (by 60 and 61.2, respectively; $p<0.0001$). CONCLUSIONS: The programed myocyte cell death triggered by ischemia-reperfusion injury is attenuated by FGF-1 in its native or non mitogenic isoforms, suggesting that this effect does not depend on the mitogenic properties of this protein. FGF-1 would contribute to the functional preservation of the myocardium after acute myocardial infarction.

PMID: 9385115 [PubMed - indexed for MEDLINE]

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1: Clin Cardiol. 1997 Nov;20(11 Suppl 2):II-52-7. Related Articles, Links

Growth factors as a potential new treatment for ischemic heart disease.

Bauters C.

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Growth factors such as fibroblast growth factor (FGF) and vascular endothelial growth factor (VEGF) exert important effects on endothelial cells in vitro and in vivo. This article reviews the effect of these two growth factors on endothelial dysfunction in various animal models of vascular disease: (1) collateral circulation supplying an ischemic territory, (2) balloon injury, and (3) diet-induced experimental atherosclerosis. Endothelial dysfunction may limit the beneficial effects of collateral vessels on tissue perfusion. Administration of VEGF or basic FGF (bFGF) augments collateral development in different models of hindlimb ischemia by enhancing neovascularity and by facilitating the recovery of endothelial function in the collateral circulation. Similarly, studies performed after balloon angioplasty have demonstrated abnormal responses of previously dilated sites to endothelium-dependent agonists. Administration of VEGF or bFGF increases endothelial regrowth and normalizes endothelium-dependent responses after experimental angioplasty. Finally, endothelium-dependent relaxation is impaired in diet-induced experimental atherosclerosis. It was recently demonstrated that hypercholesterolemic rabbits treated with bFGF had significantly better endothelium-dependent responses than those not treated with bFGF. These results show that in vivo administration of the endothelial cell growth factors VEGF and bFGF leads to significant improvement in endothelium-dependent responses and supports the concept of using these growth factors as a new therapeutic strategy for patients with vascular diseases.

Publication Types:
- Review

1: Nat Med. 1996 May;2(5):534-9. Related Articles, Links

Comment in:
- Nat Med. 1996 May;2(5):519-20.

Intracoronary gene transfer of fibroblast growth factor-5 increases blood flow and contractile function in an ischemic region of the heart.

Giordano FJ, Ping P, McKirnan MD, Nozaki S, DeMaria AN, Dillmann WH, Mathieu-Costello O, Hammond HK.

Department of Medicine, Veteran's Affairs Medical Center-San Diego, California, USA.

Increased coronary blood vessel development could potentially benefit patients with ischemic heart disease. In a model of stress-induced myocardial ischemia, intracoronary injection of a recombinant adenovirus expressing human fibroblast growth factor-5 (FGF-5) resulted in messenger RNA and protein expression of the transferred gene. Two weeks after gene transfer, regional abnormalities in stress-induced function and blood flow were improved, effects that persisted for 12 weeks. Improved blood flow and function were associated with evidence of angiogenesis. This report documents, for the first time, successful amelioration of abnormalities in myocardial blood flow and function following in vivo gene transfer.

PMID: 8616711 [PubMed - indexed for MEDLINE]

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1: J Mol Med. 1995 Jul;73(7):333-46. Related Articles, Links

Angiogenesis: mechanistic insights, neovascular diseases, and therapeutic prospects.

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This review of angiogenesis aims to describe (a) stimuli that either elicit or antagonize angiogenesis, (b) the response of the vasculature to angiogenic or anti-angiogenic stimuli, i.e., processes required for the formation of new vessels, (c) aspects of angiogenesis relating to tissue remodeling and disease, and (d) the potential of angiogenic or antiangiogenic therapeutic measures. Angiogenesis, the formation of new vessels from existing microvessels, is important in embryogenesis, wound healing, diabetic retinopathy, tumor growth, and other diseases. Hypoxia and other as yet ill-defined stimuli drive tumor, inflammatory, and connective tissue cells to generate angiogenic molecules such as vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), transforming growth factor-beta (TGF-beta), platelet-derived growth factor (PDGF), and others. Natural and synthetic angiogenesis inhibitors such as angiostatin and thalidomide can repress angiogenesis. Angiogenic and antiangiogenic molecules control the formation of new vessels via different mechanisms. VEGF and FGF elicit their effects mainly via direct action on relevant endothelial cells. TGF-beta and PDGF can attract inflammatory or connective tissue cells which in turn control angiogenesis. Additionally, PDGF may act differently on specific phenotypes of endothelial cells that are engaged in angiogenesis or that are of microvascular origin. Thus phenotypic traits of endothelial cells committed to angiogenesis may determine their cellular responses to given stimuli. Processes necessary for new vessel formation and regulated by angiogenic/antiangiogenic molecules include the migration and proliferation of endothelial cells from the microvasculature, the controlled expression of proteolytic enzymes, the breakdown and reassembly of extracellular matrix, and the

morphogenic process of endothelial tube formation. In animal models some angiogenesis-dependent diseases can be controlled via induction or inhibition of new vessel formation. Life-threatening infantile hemangiomas are a first established indication for antiangiogenic therapy in humans. Treatment of other diseases by modulation of angiogenesis are currently tested in clinical trials. Thus the manipulation of new vessel formation in angiogenesis-dependent conditions such as wound healing, inflammatory diseases, ischemic heart and peripheral vascular disease, myocardial infarction, diabetic retinopathy, and cancer is likely to create new therapeutic options.

Publication Types:

- Review

PMID: 8520966 [PubMed - indexed for MEDLINE]
